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NEWS 4 JAN 16 IPC version 2007.01 thesaurus available on STN
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NEWS 6 JAN 22 CA/CAPLUS updated with revised CAS roles
NEWS 7 JAN 22 CA/CAPLUS enhanced with patent applications from India
NEWS 8 JAN 29 PHAR reloaded with new search and display fields
NEWS 9 JAN 29 CAS Registry Number crossover limit increased to 300,000 in
multiple databases
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NEWS 17 FEB 26 CAS Registry Number crossover limit increased from 10,000
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NEWS 18 MAR 15 WPIDS/WPIX enhanced with new FRAGHITSTR display format
NEWS 19 MAR 16 CASREACT coverage extended
NEWS 20 MAR 20 MARPAT now updated daily
NEWS 21 MAR 22 LWPI reloaded
NEWS 22 MAR 30 RDISCLOSURE reloaded with enhancements
NEWS 23 MAR 30 INPADOCDB will replace INPADOC on STN
NEWS 24 APR 02 JICST-EPLUS removed from database clusters and STN

NEWS EXPRESS NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT
MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.

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FILE LAST UPDATED: 29 Apr 2007 (20070429/ED)

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=> s inducible nitric oxide synthase

64793 INDUCIBLE

186014 NITRIC

3 NITRICS

186017 NITRIC

(NITRIC OR NITRICS)

1752238 OXIDE

356029 OXIDES

1852864 OXIDE

(OXIDE OR OXIDES)

103797 SYNTHASE

5996 SYNTHASES

104884 SYNTHASE

(SYNTHASE OR SYNTHASES)

L1 8934 INDUCIBLE NITRIC OXIDE SYNTHASE

(INDUCIBLE(W)NITRIC(W)OXIDE(W)SYNTHASE)

=> s l1 and inflammator?

177669 INFLAMMATOR?

L2 2688 L1 AND INFLAMMATOR?

=> s l2 and diseases?

1070477 DISEAS?

L3 1470 L2 AND DISEAS?

=> s l3 and central nervous system

401077 CENTRAL

29 CENTRALS

401100 CENTRAL

(CENTRAL OR CENTRALS)

217702 NERVOUS

2425212 SYSTEM

1328273 SYSTEMS

3287324 SYSTEM
(SYSTEM OR SYSTEMS)
81625 CENTRAL NERVOUS SYSTEM
(CENTRAL(W)NERVOUS(W)SYSTEM)
L4 90 L3 AND CENTRAL NERVOUS SYSTEM

=> s l4 and py<2003
22885160 PY<2003
L5 40 L4 AND PY<2003

=> d ibib abs hitstr 1-10

L5 ANSWER 1 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:899394 CAPLUS

DOCUMENT NUMBER: 138:378830

TITLE: Inhibitors of poly(ADP-ribose) polymerase-1 suppress transcriptional activation in lymphocytes and ameliorate autoimmune encephalomyelitis in rats

AUTHOR(S): Chiarugi, Alberto

CORPORATE SOURCE: Department of Preclinical and Clinical Pharmacology, University of Florence, Florence, Italy

SOURCE: British Journal of Pharmacology (2002), 137(6), 761-770

CODEN: BJPCBM; ISSN: 0007-1188

PUBLISHER: Nature Publishing Group

DOCUMENT TYPE: Journal

LANGUAGE: English

AB In the presence of genotoxic stress poly(ADP-ribose) polymerase-1 (PARP-1) leads to NAD⁺ and ATP depletion, participating in the pathogenesis of several disorders including inflammation. Accordingly, chemical inhibitors of PARP-1 are efficacious anti-inflammatories, albeit the underlying mol. mechanisms are still under debate. This study investigated the effect of the PARP-1 inhibitors 6(5H)-phenanthridinone and benzamide as well as that of benzoic acid, an inactive analog of benzamide, on development of exptl. allergic encephalomyelitis (EAE) in rats. Both 6(5H)-phenanthridinone and benzamide attenuated development of EAE, reducing clin. score, neuroimmune infiltration and expression of inflammatory mediators such as inducible nitric oxide synthase, interleukin-1 β and -2, cyclooxygenase-2, tumor necrosis factor- α and interferon- γ in the spinal cord of myelin-immunized rats. Importantly, no evidence of NAD⁺ and ATP depletion as well as poly(ADP-ribose) formation was detected in the spinal cord. By contrast, a robust formation of poly(ADP-ribose) occurred in B- and T-cell areas in lymph nodes of myelin-immunized rats and was suppressed by the treatment with 6(5H)-phenanthridinone and benzamide. In cultures of activated rat lymphocytes, 6(5H)-phenanthridinone and benzamide reduced the DNA-binding activity of NF- κ B and AP-1 and transcription of pro-inflammatory cytokines such as interleukin-2, interferon- γ and tumor necrosis factor- α . Notably, benzoic acid did not reproduce the in vivo and in vitro effects of its parent compound. These findings indicate that PARP-1 promotes transcriptional activation in lymphocytes and inhibitors of its enzymic activity are useful for the treatment of autoimmune disorders of the central nervous system.

REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 2 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:741590 CAPLUS

DOCUMENT NUMBER: 138:34588

TITLE: Ethanol-Induced Modulation of Inducible Nitric Oxide Synthase Activity in Human A172 Astrocytoma Cells

AUTHOR(S): Davis, Randall L.; Dertien, Janet; Syapin, Peter J.
CORPORATE SOURCE: Alcohol and Brain Research Laboratory, Department of
Pharmacology, Texas Tech University Health Sciences
Center, Lubbock, TX, USA
SOURCE: Alcoholism: Clinical and Experimental Research (2002), 26(9), 1404-1411
CODEN: ACRSDM; ISSN: 0145-6008
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Background: Glial cells are critical in the functioning of the central nervous system (CNS), including responsiveness to injury and immunocompetence. The immune and inflammatory response involves the inducible form of nitric oxide synthase (iNOS), and subsequent NO production. Previously, the authors have demonstrated that ethanol inhibits cytokine-induced iNOS expression and activity in rat glial cells. Evidence of ethanol-induced effects on iNOS in human glial cells is nonexistent. Herein, the conditions necessary for significant iNOS induction in human A172 astrocytoma cells have been characterized, and subsequently, the effects of ethanol on iNOS expression have been investigated. Methods: A172 cells were analyzed immunohistochem. for the astrocyte markers, glial fibrillary acidic protein (GFAP) and S-100 β . The ability of A172 cells to express iNOS was assessed by stimulating cells with interferon- γ (IFN γ), tumor necrosis factor- α (TNF α), interleukin-1 β (IL-1 β), bacterial lipopolysaccharide (LPS), L-arginine, and tetrahydrobiopterin (BH4) in various combinations. Following stimulation, iNOS induction was monitored via measurement of nitrite production and in vitro iNOS enzyme activity. Time-course (6-24 h) studies assessed the effects of ethanol (50-200 mM) on iNOS induction. Results: Immunohistochem. anal. confirmed that A172 cells were phenotypically, astrocytic. Induction of nitrite production by a cytotoxic mix [IFN γ (100 ng/mL) + TNF α (30 ng/mL) + IL-1 β (5 ng/mL)] was differentially enhanced by exposure to supplemental factors including LPS, L-arginine, and BH4. Nitrite production was greatest over the initial 24 h of stimulation with iNOS enzyme activity peaking at 12 h. Acute (6-24 h) exposure of activated cells to 50 mM ethanol enhanced iNOS activity recovered from the cytosol, whereas 200 mM ethanol decreased it. Ethanol had no direct effect on the catalytic activity of the enzyme. Conclusions: The present study is the first published report of ethanol-induced modulation of iNOS expression in human glial cells. The data suggest that ethanol is influencing iNOS enzyme levels most profoundly. Altered astrocyte function may be a point of ethanol-induced perturbation in CNS immune function. These findings should lend insight into the role of ethanol on human CNS immunity and brain injury.

REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:719058 CAPLUS
DOCUMENT NUMBER: 138:3199
TITLE: M-CSF deficiency leads to reduced metallothioneins I and II expression and increased tissue damage in the brain stem after 6-aminonicotinamide treatment
AUTHOR(S): Penkowa, Milena; Poulsen, Christian Bjorn; Carrasco, Javier; Hidalgo, Juan
CORPORATE SOURCE: Department of Medical Anatomy, The Panum Institute, University of Copenhagen, Copenhagen, DK-2200, Den.
SOURCE: Experimental Neurology (2002), 176(2), 308-321
CODEN: EXNEAC; ISSN: 0014-4886
PUBLISHER: Elsevier Science
DOCUMENT TYPE: Journal

LANGUAGE: English

AB 6-Aminonicotinamide (6-AN) is a niacin antagonist, which leads to degeneration of gray-matter astrocytes followed by a vigorous inflammatory response. Macrophage colony stimulating factor (M-CSF) is important during inflammation, and to further clarify the roles for M-CSF in neurodegeneration and brain cell death, the authors have examined the effect of 6-AN on osteopetrotic mice with genetic M-CSF deficiency (op/op mice). The 6-AN-induced degeneration of graymatter areas was comparable in control and op/op mice, but the nos. of reactive astrocytes, macrophages, and lymphocytes in the damaged areas were significantly decreased in op/op mice relative to controls. The levels of oxidative stress (as determined by using immunoreactivity for inducible nitric oxide synthase, nitrotyrosine, and malondialdehyde) and apoptotic cell death (as determined by using TUNEL and immunoreactivity for caspases and cytochrome c) were significantly increased in 6-AN-injected op/op mice relative to controls. From a number of antioxidant factors assayed, only metallothioneins I and II (MT-I+II) were decreased in op/op mice in comparison to controls. Thus, the present results indicate that M-CSF is an important growth factor for coping with 6-AN-induced central nervous system damage and suggest that MT-I+II are likely to have a significant role.

REFERENCE COUNT: 93 THERE ARE 93 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 4 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:694982 CAPLUS

DOCUMENT NUMBER: 138:265333

TITLE: Discovery of new chemical classes of synthetic ligands that suppress neuroinflammatory responses

AUTHOR(S): Watterson, D. Martin; Haiech, Jacques; Van Eldik, Linda J.

CORPORATE SOURCE: Drug Discovery Program and Departments of Molecular Pharmacology and Biological Chemistry, Northwestern University Medical School, Chicago, IL, 60611-3008, USA

SOURCE: Journal of Molecular Neuroscience (2002), 19(1/2), 89-93

CODEN: JMNEES; ISSN: 0895-8696

PUBLISHER: Humana Press Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The authors used a chemical genomics approach that includes follow up in parallel syntheses to discover a new class of compds. that selectively suppress glial activation. While the mechanism of action remains to be determined, available data and the exptl. approach for discovery indicate that the mechanism includes inhibition of gene regulating protein kinases. Specifically, the increased production of IL-1 β and iNOS in response to various activating stimuli, including A β 1-42, is suppressed while the production of potentially beneficial responses, such as ApoE production, is not inhibited. The increased production of COX-2 and p38 MAPK activation are also not altered, demonstrating the novel nature of potential therapeutic targets compared to currently available drugs. The chemical scaffold is 3-aminopyridazine (3-AP). This is an attractive scaffold because of its potential for diversification by established, facile chemistries and the prior use of a 3-AP scaffold in other central nervous system targeted therapeutics. Therefore, the potential bioavailability of 3-AP derivs. and the demonstrated cellular selectivity demand that future research address the potential efficacy of selective 3-AP derivs. in animal models of disease.

REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 5 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:594628 CAPLUS
 DOCUMENT NUMBER: 137:150265
 TITLE: Substituted aryl compounds as novel cyclooxygenase-2 selective inhibitors, compositions and methods of use
 INVENTOR(S): Khanapure, Subhash P.; Garvey, David S.; Earl, Richard A.; Ezawa, Maiko; Fang, Xinqin; Gaston, Ricky D.
 PATENT ASSIGNEE(S): Nitromed, Inc., USA
 SOURCE: PCT Int. Appl., 132 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|------|----------|-----------------|--------------|
| WO 2002060378 | A2 | 20020808 | WO 2001-US48823 | 20011221 <-- |
| WO 2002060378 | A3 | 20031231 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| CA 2432642 | A1 | 20020808 | CA 2001-2432642 | 20011221 <-- |
| AU 2002249812 | A1 | 20020812 | AU 2002-249812 | 20011221 <-- |
| US 2002119977 | A1 | 20020829 | US 2001-24046 | 20011221 <-- |
| US 6706724 | B2 | 20040316 | | |
| EP 1406609 | A2 | 20040414 | EP 2001-998052 | 20011221 |
| EP 1406609 | B1 | 20060906 | | |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR | | | | |
| JP 2005502587 | T | 20050127 | JP 2002-560574 | 20011221 |
| AT 338544 | T | 20060915 | AT 2001-998052 | 20011221 |
| US 2004116431 | A1 | 20040617 | US 2003-730979 | 20031210 |
| US 6825185 | B2 | 20041130 | | |
| US 2005059665 | A1 | 20050317 | US 2004-969079 | 20041021 |
| PRIORITY APPLN. INFO.: | | | | |
| | | | US 2000-256932P | P 20001221 |
| | | | US 2001-24046 | A3 20011221 |
| | | | WO 2001-US48823 | W 20011221 |
| | | | US 2003-730979 | A1 20031210 |

OTHER SOURCE(S): MARPAT 137:150265

AB Substituted aryl compds. that are cyclooxygenase 2 (COX-2) selective inhibitors and compns. comprising at least one COX-2 selective inhibitor, and, optionally, at least one compound that donates, transfers or releases nitric oxide, stimulates endogenous synthesis of nitric oxide, elevates endogenous levels of endothelium-derived relaxing factor or is a substrate for nitric oxide synthase, and/or, optionally, at least one therapeutic agent are described. A therapeutic agent is selected from steroids, nonsteroidal anti-inflammatory compds. (NSAID), 5-lipoxygenase (5-LO) inhibitors, leukotriene B4 (LTB4) receptor antagonists, leukotriene A4 (LTA4) hydrolase inhibitors, 5-HT agonists, 3-hydroxy-3-methylglutaryl CoA (HMG-CoA) inhibitors, H2 antagonists, antineoplastic agents, antiplatelet agents, thrombin inhibitors, thromboxane inhibitors, decongestants, diuretics, sedating or non-sedating antihistaminics, inducible nitric oxide synthase inhibitors, opioids, analgesics, Helicobacter pylori inhibitors, proton pump inhibitors, and isoprostane inhibitors. The invention also provides novel kits comprising at least one COX-2 selective inhibitor, and,

optionally, at least one nitric oxide donor, and/or, optionally, at least one therapeutic agent. The cyclooxygenase-2 selective inhibitors of the invention can be optionally nitrosated and/or nitrosylated. The invention also provides methods for treating inflammation, pain and fever; for treating and/or improving the gastrointestinal properties of COX-2 selective inhibitors; for facilitating wound healing; for treating and/or preventing renal toxicity or other toxicities; for treating and/or preventing other disorders resulting from elevated levels of cyclooxygenase-2; and for improving the cardiovascular profile of COX-2 selective inhibitors.

L5 ANSWER 6 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:458947 CAPLUS

DOCUMENT NUMBER: 137:123894

TITLE: Intracerebroventricular but not intravenous interleukin-1 β induces widespread vascular-mediated leukocyte infiltration and immune signal mRNA expression followed by brain-wide glial activation

AUTHOR(S): Proescholdt, M. G.; Chakravarty, S.; Foster, J. A.; Foti, S. B.; Briley, E. M.; Herkenham, M.

CORPORATE SOURCE: Section on Functional Neuroanatomy, National Institute of Mental Health, Bethesda, MD, 20892-4070, USA

SOURCE: Neuroscience (Oxford, United Kingdom) (2002), 112(3), 731-749

CODEN: NRSCDN; ISSN: 0306-4522

PUBLISHER: Elsevier Science Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Interleukin-1 β (IL-1 β) is a pro-inflammatory cytokine that appears in brain and cerebrospinal fluid following peripheral immune challenges and central infections or injury. The authors examined the consequences of i.c.v. infusion of IL-1 β on mRNA expression of several immune markers and on recruitment of peripheral leukocytes. Awake rats were infused with IL-1 β (100 ng/rat) into the lateral ventricle, and 0.5, 2, 4, 8, 12, or 24 h later, animals were killed and their fresh-frozen brains processed for in situ hybridization and immunohistochem. Widespread vascular expression of inhibitory factor κ B α (I κ B α , marker of nuclear factor κ B α transcriptional activity) and inducible cyclooxygenase (COX-2) mRNAs at 0.5-2 h was credited to movement of IL-1 β along ventricular, subarachnoid, and perivascular pathways to target endothelia that express type 1 IL-1 receptor mRNA. Induction of monocyte chemoattractant protein-1 mRNA and intercellular adhesion mol.-1 (ICAM-1) immunostaining on endothelia began at 0.5-2 h. Leukocytes (neutrophils and monocytes, recognized by morphol. and CD45 and ED1 immunostaining) appeared in meninges and blood vessels at 2-4 h and diffusely penetrated the parenchyma at 8-24 h. The leukocytes strongly expressed IL-1 β and inducible nitric oxide synthase mRNAs. Beginning at 4-12 h, astrocytes (glial acidic fibrillary protein mRNA and protein and c-fos mRNA) and microglia (ionized calcium-binding adaptor mol. 1 mRNA and protein) showed widespread activation. Other rats received i.v. IL-1 β (6 μ g/kg). Their brains showed induction of I κ B α and COX-2 mRNAs in the vasculature at 2 h but none of the other sequelae. In summary, the data indicate that IL-1 β in the cerebrospinal fluid reaches its target receptors on the endothelia via perivascular volume transmission, up-regulates ICAM-1, and triggers a targeted leukocyte emigration and widespread glial activation stimulated perhaps by pro-inflammatory mols. expressed by leukocytes. The dramatic difference between i.c.v. and i.v. routes of administration underscores the potency of IL-1 β within the brain to dynamically affect the cellular trafficking component of 'immune privilege'.

REFERENCE COUNT: 68 THERE ARE 68 CITED REFERENCES AVAILABLE FOR THIS

L5 ANSWER 7 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:382961 CAPLUS

DOCUMENT NUMBER: 137:62013

TITLE: Role of mitogen-activated protein kinases in inducible nitric oxide synthase and TNF α expression in human fetal astrocytes

AUTHOR(S): Hua, Liwei L.; Zhao, Meng-Liang; Cosenza, Melissa; Kim, Mee-Ohk; Huang, Huan; Tanowitz, Herbert B.; Brosnan, Celia F.; Lee, Sunhee C.

CORPORATE SOURCE: Department of Pathology, Albert Einstein College of Medicine, Bronx, NY, 10461, USA

SOURCE: Journal of Neuroimmunology (2002), 126(1-2), 180-189

CODEN: JNRIDW; ISSN: 0165-5728

PUBLISHER: Elsevier Science B.V.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Astrocytes are important sources of proinflammatory mediators such as iNOS and TNF α in the diseased central nervous system. In previous studies, we showed that the cytokine IL-1 plays a critical role in the activation of human astrocytes to express TNF α and the inducible form of nitric oxide synthase (iNOS). In the present study, we have addressed the role of the MAP-kinase pathway in the signaling events leading to the induction of these genes. Treatment with SB203580, a specific inhibitor of p38 mitogen-activated protein kinases (MAPK), potently inhibited IL-1-mediated induction of iNOS and TNF α in cultures of human fetal astrocytes. In contrast, PD98059, an upstream inhibitor of the extracellular regulated kinase (ERK)1/2 pathway, had little or no effect. Interestingly, SB203580 reduced the mRNA expression for iNOS, TNF α , and IL-6, indicating inhibition prior to translation. Transfection of astrocytes with a dominant-neg. Jun-NH2-terminal kinase (JNK) construct also reduced iNOS expression. Western blot anal. showed phosphorylated p38 and JNK in IL-1-activated astrocytes, and phosphorylated ERK in both resting and activated cells. Electrophoretic mobility shift assay (EMSA) showed that IL-1 induced NF- κ B and AP-1 DNA complex formation in astrocytes, and that SB203580 inhibited AP-1 complex formation. These results demonstrate the differential roles played by the three MAP kinases in human astrocyte inflammatory gene activation and point to a crucial function of p38 and JNK MAP kinases in IL-1-mediated astrocyte activation.

REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 8 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:366268 CAPLUS

DOCUMENT NUMBER: 137:199764

TITLE: Identification of new therapeutic targets for prevention of CNS inflammation

AUTHOR(S): Owens, Trevor

CORPORATE SOURCE: Neuroimmunology Unit, Montreal Neurological Institute, Montreal, QC, H3A 2B4, Can.

SOURCE: Expert Opinion on Therapeutic Targets (2002), 6(2), 203-215

CODEN: EOTTAO; ISSN: 1472-8222

PUBLISHER: Ashley Publications Ltd.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Multiple sclerosis (MS) is a disease of complex pathologies, which involves infiltration by CD4+ and CD8+ T cells of and response within the central nervous system.

Expression in the CNS of cytokines, reactive nitrogen species and costimulator mols. have all been described in MS. Notably, the cytokines IFN- γ and TNF are strongly expressed. Microglial cells in the CNS express costimulator mols. and it is assumed that they play a role in directing or inducing the T cell response. Transgenic expts. have tested the effects of overexpression of these mols. in mice and have shown that TNF has multiple effects in the CNS. These range from pro-inflammatory effects of soluble TNF signalling through one of its receptors TNF-RI, to protective/regenerative effects of membrane-associated TNF signalling through the other receptor, TNF-RII. Although IFN- γ induces nitric oxide production via the enzyme inducible nitric oxide synthase, which is immunosuppressive, IFN- γ is predominantly pro-inflammatory. In CNS disease in mice that involves CD8+ T cells, IFN- γ blockade is protective. Finally, microglial expression of the costimulator ligand B7.2 induces demyelinating pathol. Animal expts. therefore point to IFN- γ and costimulatory microglia as logical targets of therapy for MS. IFN- γ represents a more accessible target and should therefore be pursued at the earliest opportunity.

REFERENCE COUNT: 89 THERE ARE 89 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 9 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:294227 CAPLUS

DOCUMENT NUMBER: 136:315023

TITLE: Polydithiocarbamate-containing nontargeting macromolecules for therapeutic and diagnostic applications

INVENTOR(S): Lai, Ching-san

PATENT ASSIGNEE(S): Medinox, Inc., USA

SOURCE: U.S. Pat. Appl. Publ., 18 pp., Cont.-in-part of U.S. Ser. No. 899,087, abandoned.

CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|------------------------|------|----------|-----------------|--------------|
| US 2002045573 | A1 | 20020418 | US 1999-409645 | 19991001 <-- |
| US 6649591 | B2 | 20031118 | | |
| CN 1230178 | A | 19990929 | CN 1997-197797 | 19970828 <-- |
| KR 2000035992 | A | 20000626 | KR 1999-701945 | 19990309 <-- |
| PRIORITY APPLN. INFO.: | | | US 1996-25867P | P 19960910 |
| | | | US 1997-899087 | B2 19970723 |

OTHER SOURCE(S): MARPAT 136:315023

AB A new class of drugs for treatment of such indications as cerebral stroke and other ischemia/reperfusion injury is disclosed. Dithiocarbamates are linked to the surface of a non-immunogenic, nontargeting macromol. other than an antibody (e.g., albumin) either by using crosslinking reagents or by nonspecific binding to produce polydithiocarbamate-macromol.-containing comps., which represent a new class of drugs for treatment of such indications as cerebral stroke and other ischemia/reperfusion injury. Combinational therapeutic methods have been developed for the in vivo inactivation or inhibition of formation (either directly or indirectly) of species which induce the expression of inducible nitric oxide synthase, as well as reducing nitric oxide levels produced as a result of NO synthase expression. Magnetic resonance imaging methods have been developed for the measurement of cerebral and cardiac blood flow and infarct volume in ischemic stroke or heart attack situations. Such methods employ iron-containing complexes of a composition comprising a dithiocarbamate and a nonimmunogenic, nontargeting macromol.

other than an antibody as contrast agents. The crosslinking expts. were performed by the treatment of bovine serum albumin (BSA) with N-hydroxysulfosuccinimidyl-4-azido salicylic acid in DMSO at pH 7.0. The reaction mixture was incubated at ambient temperature for 10-60 min. Upon the addition of N-methyl-D-glucamine dithiocarbamate (MGD), the solution was irradiated at 365 nm for 1-5 min. After irradiation, the solution was applied onto a G-25 pre-packed column. The MGD-BSA containing fractions were collected and rechromatographed once. The stoichiometry of MGD bound to the BSA mol. can be estimated by measuring the absorbance at 215 nm (for MGD) and 280 nm (for BSA).

L5 ANSWER 10 OF 40 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:151387 CAPLUS

DOCUMENT NUMBER: 137:134928

TITLE: Water-soluble chitosan inhibits the production of pro-inflammatory cytokine in human astrocytoma cells activated by amyloid β peptide and interleukin-1 β

AUTHOR(S): Kim, Mi-Sun; Sung, Man-Joon; Seo, Sang-Bong; Yoo, Su-Jin; Lim, Woon-Ki; Kim, Hyung-Min

CORPORATE SOURCE: Department of Oriental Pharmacy, Wonkwang University, College of Pharmacy and Korea Institute of Oriental Pharmacy, Chonbuk, Iksan, 570-749, S. Korea

SOURCE: Neuroscience Letters (2002), 321(1-2), 105-109

CODEN: NELED5; ISSN: 0304-3940

PUBLISHER: Elsevier Science Ireland Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB A chronic inflammatory response associated with β -amyloid ($A\beta$) and interleukin-1 β (IL-1 β) is responsible for the pathol. of Alzheimer's disease (AD). Astrocytes are predominant neuroglial cells of the central nervous system and are actively involved in cytokine-mediated events in AD. To investigate the biol. effect of water-soluble chitosan (WSC), we examined cytotoxicity, production of pro-inflammatory cytokines and inducible nitric-oxide synthase (iNOS) on human astrocytoma cell line CCF-STTG1 stimulated with IL-1 β and $A\beta$ fragment 25-35 ($A\beta$ [25-35]). In 3-[4,5-dimethylthiazol-2-yl]-2,5-diphenyltetrazolium bromide colorimetric assay, WSC by itself had no effect on cell viability on human astrocytoma cells. The effects of WSC on tumor necrosis factor- α (TNF- α) and interleukin-6 (IL-6) were evaluated with ELISA and Western blotting. The production of TNF- α and IL-6 was induced by IL-1 β and $A\beta$ [25-35] and synergistically amplified by the co-stimulation of IL-1 β and $A\beta$ [25-35]. The secretion and expression of pro-inflammatory cytokines, TNF- α and IL-6, was significantly inhibited by pretreatment with WSC in human astrocytoma cells. The expression of iNOS was induced by IL-1 β and $A\beta$ [25-35] and was partially inhibited by treatment with WSC. We demonstrate the regulatory effects of WSC in human astrocytes for the first time and suggest the anti-inflammatory effect of WSC may reduce and delay AD pathol. events.

REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> FIL STNGUIDE

COST IN U.S. DOLLARS

| | |
|------------|---------|
| SINCE FILE | TOTAL |
| ENTRY | SESSION |
| 50.28 | 50.49 |

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

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| SINCE FILE | TOTAL |
| ENTRY | SESSION |

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LAST RELOADED: Apr 27, 2007 (20070427/UP).

=> d his

(FILE 'HOME' ENTERED AT 10:23:11 ON 30 APR 2007)

FILE 'CAPLUS' ENTERED AT 10:23:34 ON 30 APR 2007
L1 8934 S INDUCIBLE NITRIC OXIDE SYNTHASE
L2 2688 S L1 AND INFLAMMATOR?
L3 1470 S L2 AND DISEAS?
L4 90 S L3 AND CENTRAL NERVOUS SYSTEM
L5 40 S L4 AND PY<2003

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=> log y

| | | |
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| FULL ESTIMATED COST | 0.24 | 50.73 |
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| | ENTRY | SESSION |
| CA SUBSCRIBER PRICE | 0.00 | -7.80 |

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NEWS 8 JAN 29 PHAR reloaded with new search and display fields
NEWS 9 JAN 29 CAS Registry Number crossover limit increased to 300,000 in
multiple databases
NEWS 10 FEB 15 PATDPASPC enhanced with Drug Approval numbers
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NEWS 15 FEB 26 TOXCENTER enhanced with reloaded MEDLINE
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NEWS 17 FEB 26 CAS Registry Number crossover limit increased from 10,000
to 300,000 in multiple databases
NEWS 18 MAR 15 WPIDS/WPIX enhanced with new FRAGHITSTR display format
NEWS 19 MAR 16 CASREACT coverage extended
NEWS 20 MAR 20 MARPAT now updated daily
NEWS 21 MAR 22 LWPI reloaded
NEWS 22 MAR 30 RDISCLOSURE reloaded with enhancements
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NEWS EXPRESS NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT
MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.

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| ENTRY | SESSION |
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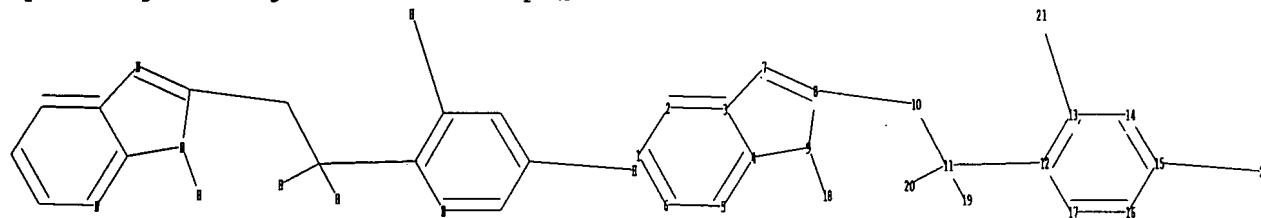
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<http://www.cas.org/support/stngen/stdoc/properties.html>

Uploading C:\Program Files\Stnexp\Queries\10573204a.str



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ring nodes :
1 2 3 4 5 6 7 8 9 12 13 14 15 16 17
chain bonds :
8-10 9-18 10-11 11-12 11-19 11-20 13-21 15-22
ring bonds :
1-2 1-6 2-3 3-4 3-7 4-5 4-9 5-6 7-8 8-9 12-13 12-17 13-14 14-15 15-16
16-17
exact/norm bonds :
3-7 4-9 7-8 8-9
exact bonds :
8-10 9-18 10-11 11-12 11-19 11-20 13-21 15-22
normalized bonds :
1-2 1-6 2-3 3-4 4-5 5-6 12-13 12-17 13-14 14-15 15-16 16-17
isolated ring systems :
containing 1: 12 :

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G1:C,H

Match level :

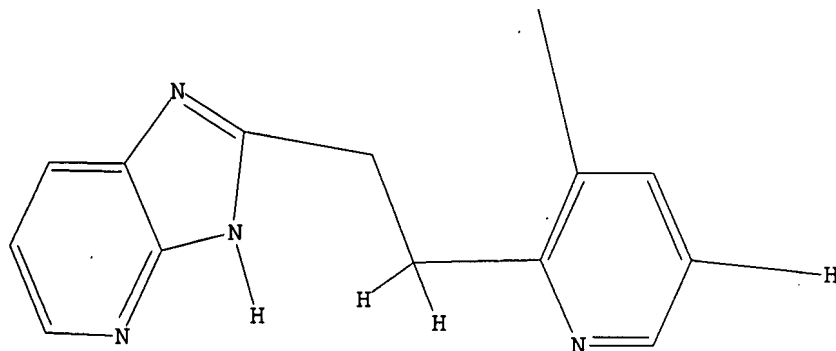
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11:CLASS 12:Atom 13:Atom 14:Atom 15:Atom 16:Atom 17:Atom 18:CLASS 19:CLASS
20:CLASS 21:CLASS 22:CLASS

L1 STRUCTURE UPLOADED

=> d l1

L1 HAS NO ANSWERS

L1 STR



G1 C,H

Structure attributes must be viewed using STN Express query preparation.

=> s l1

SAMPLE SEARCH INITIATED 10:00:14 FILE 'REGISTRY'
SAMPLE SCREEN SEARCH COMPLETED - 38 TO ITERATE

100.0% PROCESSED 38 ITERATIONS 5 ANSWERS
SEARCH TIME: 00.00.01

FULL FILE PROJECTIONS: ONLINE **COMPLETE**
BATCH **COMPLETE**
PROJECTED ITERATIONS: 391 TO 1129
PROJECTED ANSWERS: 5 TO 234

L2 5 SEA SSS SAM L1

=> s l1 full

FULL SEARCH INITIATED 10:00:19 FILE 'REGISTRY'
FULL SCREEN SEARCH COMPLETED - 769 TO ITERATE

100.0% PROCESSED 769 ITERATIONS 161 ANSWERS
SEARCH TIME: 00.00.01

L3 161 SEA SSS FUL L1

=> file caplus

COST IN U.S. DOLLARS

SINCE FILE
ENTRY

TOTAL
SESSION

FULL ESTIMATED COST

172.10

172.31

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FILE COVERS 1907 - 30 Apr 2007 VOL 146 ISS 19
FILE LAST UPDATED: 29 Apr 2007 (20070429/ED)

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=> s 13 full
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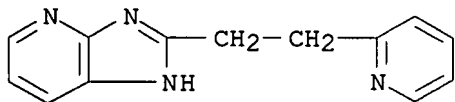
=> s 19 and py<2003
L9 NOT FOUND
The L-number entered could not be found. To see the definition of L-numbers, enter DISPLAY HISTORY at an arrow prompt (=>).

=> s 14 and py<2003
22885160 PY<2003
L5 1 L4 AND PY<2003

=> d ibib abs hitstr tot

L5 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1999:284034 CAPLUS
DOCUMENT NUMBER: 131:82668
TITLE: Synthesis and antiproliferative activity of some N-sulfonated-2-substituted benzimidazoles and imidazo[4,5-b]pyridines
AUTHOR(S): Garuti, Laura; Varoli, Lucilla; Cermelli, Claudio; Baggio, Giosue; Lupo, Lucia; Malagoli, Monica; Castelli, Mario
CORPORATE SOURCE: Department of Pharmaceutical Science, University of Bologna, Bologna, I-40126, Italy
SOURCE: Anti-Cancer Drug Design (1998), 13(8), 969-980
CODEN: ACDDEA; ISSN: 0266-9536
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Some N-sulfonated-2-substituted benzimidazoles and imidazo[4,5-b]-pyridines were synthesized and tested in vitro for antiviral and antiproliferative activity. None of the compds. had antiviral properties. However, 3 of them inhibited the proliferation of leukemia and lymphoma cell lines at micromolar concns. The maximum potency of antiproliferative activity is correlated with the presence of an ethylenic spacer between

the 2 heterocycles.
 IT 229468-73-9P
 RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT
 (Reactant or reagent)
 (synthesis and antiproliferative activity of N-sulfonated-2-substituted
 benzimidazoles and imidazo[4,5-b]pyridines)
 RN 229468-73-9 CAPLUS
 CN 1H-Imidazo[4,5-b]pyridine, 2-[2-(2-pyridinyl)ethyl]- (9CI) (CA INDEX
 NAME)



REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS
 RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> FIL STNGUIDE

| | | |
|--|------------|---------|
| COST IN U.S. DOLLARS | SINCE FILE | TOTAL |
| | ENTRY | SESSION |
| FULL ESTIMATED COST | 7.75 | 180.06 |
| DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) | SINCE FILE | TOTAL |
| | ENTRY | SESSION |
| CA SUBSCRIBER PRICE | -0.78 | -0.78 |

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=> file reg

| | | |
|--|------------|---------|
| COST IN U.S. DOLLARS | SINCE FILE | TOTAL |
| | ENTRY | SESSION |
| FULL ESTIMATED COST | 0.12 | 180.18 |
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| | ENTRY | SESSION |
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 DICTIONARY FILE UPDATES: 27 APR 2007 HIGHEST RN 933069-51-3

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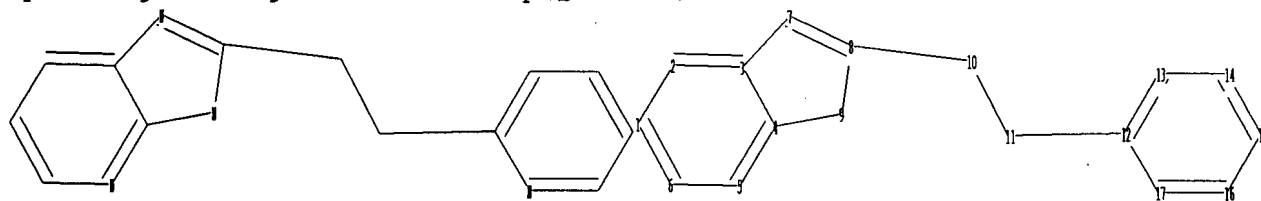
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ring nodes :

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chain bonds :

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ring bonds :

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exact/norm bonds :

3-7 4-9 7-8 8-9

exact bonds :

8-10 10-11 11-12

normalized bonds :

1-2 1-6 2-3 3-4 4-5 5-6 12-13 12-17 13-14 14-15 15-16 16-17

isolated ring systems :

containing 1 : 12 :

G1:C,H

Match level :

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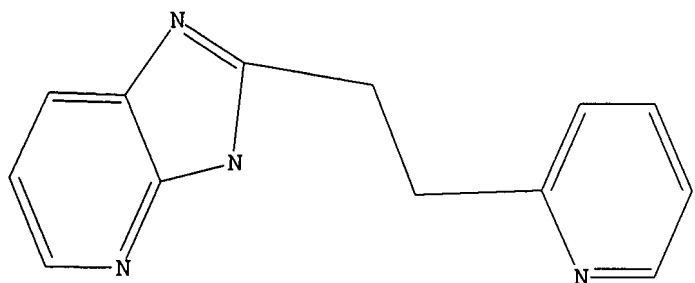
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L6 STRUCTURE UPLOADED

=> d 16

L6 HAS NO ANSWERS

L6 STR



G1 C,H

Structure attributes must be viewed using STN Express query preparation.

=> s 16 full

FULL SEARCH INITIATED 10:03:04 FILE 'REGISTRY'

FULL SCREEN SEARCH COMPLETED - 769 TO ITERATE

100.0% PROCESSED 769 ITERATIONS

163 ANSWERS

SEARCH TIME: 00.00.01

L7 163 SEA SSS FUL L6

=> file caplus

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

172.55

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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE

TOTAL

ENTRY

SESSION

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-0.78

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=> s 17 full

L8 10 L7

=> s 18 and py<2003
22885160 PY<2003
L9 2 L8 AND PY<2003

=> d ibib abs hitstr tot

L9 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1999:284034 CAPLUS

DOCUMENT NUMBER: 131:82668

TITLE: Synthesis and antiproliferative activity of some
N-sulfonated-2-substituted benzimidazoles and
imidazo[4,5-b]pyridines

AUTHOR(S): Garuti, Laura; Varoli, Lucilla; Cermelli, Claudio;
Baggio, Giosue; Lupo, Lucia; Malagoli, Monica;
Castelli, Mario

CORPORATE SOURCE: Department of Pharmaceutical Science, University of
Bologna, Bologna, I-40126, Italy

SOURCE: Anti-Cancer Drug Design (1998), 13(8),
969-980

CODEN: ACDDEA; ISSN: 0266-9536

PUBLISHER: Oxford University Press

DOCUMENT TYPE: Journal

LANGUAGE: English

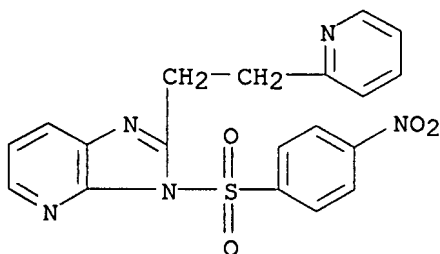
AB Some N-sulfonated-2-substituted benzimidazoles and imidazo[4,5-b]-
pyridines were synthesized and tested in vitro for antiviral and
antiproliferative activity. None of the compds. had antiviral properties.
However, 3 of them inhibited the proliferation of leukemia and lymphoma
cell lines at micromolar concns. The maximum potency of antiproliferative
activity is correlated with the presence of an ethylenic spacer between
the 2 heterocycles.

IT 229468-81-9P

RL: BAC (Biological activity or effector, except adverse); BSU (Biological
study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use);
BIOL (Biological study); PREP (Preparation); USES (Uses)
(synthesis and antiproliferative activity of N-sulfonated-2-substituted
benzimidazoles and imidazo[4,5-b]pyridines)

RN 229468-81-9 CAPLUS

CN 3H-Imidazo[4,5-b]pyridine, 3-[(4-nitrophenyl)sulfonyl]-2-[2-(2-
pyridinyl)ethyl]- (9CI) (CA INDEX NAME)



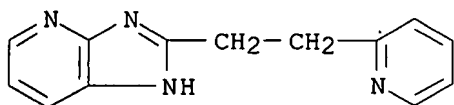
IT 229468-73-9P

RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT
(Reactant or reagent)

(synthesis and antiproliferative activity of N-sulfonated-2-substituted
benzimidazoles and imidazo[4,5-b]pyridines)

RN 229468-73-9 CAPLUS

CN 1H-Imidazo[4,5-b]pyridine, 2-[2-(2-pyridinyl)ethyl]- (9CI) (CA INDEX
NAME)



REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1997:549356 CAPLUS

DOCUMENT NUMBER: 127:152950

TITLE: Multiple unit effervescent dosage forms comprising proton pump inhibitor

INVENTOR(S): Lundberg, Per Johan

PATENT ASSIGNEE(S): Astra Aktiebolag, Swed.; Lundberg, Per Johan

SOURCE: PCT Int. Appl., 56 pp.

CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|------|----------|-----------------|--------------|
| WO 9725030 | A1 | 19970717 | WO 1996-SE1738 | 19961220 <-- |
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| RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG | | | | |
| SE 9600073 | A | 19970709 | SE 1996-73 | 19960108 <-- |
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| CA 2214027 | A1 | 19970717 | CA 1996-2214027 | 19961220 <-- |
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| BR 9607367 | A | 19971230 | BR 1996-7367 | 19961220 <-- |
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| HU 200000976 | A2 | 20001128 | HU 2000-976 | 19961220 <-- |
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| IL 121653 | A | 20010808 | IL 1996-121653 | 19961220 <-- |
| AT 251451 | T | 20031015 | AT 1996-944727 | 19961220 |
| PT 814783 | T | 20040227 | PT 1996-944727 | 19961220 |
| ES 2208775 | T3 | 20040616 | ES 1996-944727 | 19961220 |
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| US 6132770 | A | 20001017 | US 1997-793077 | 19970213 <-- |
| NO 9704051 | A | 19971015 | NO 1997-4051 | 19970903 <-- |
| NO 319999 | B1 | 20051010 | | |

PRIORITY APPLN. INFO.: SE 1996-73 A 19960108
WO 1996-SE1738 W 19961220

OTHER SOURCE(S): MARPAT 127:152950

AB A new tabletted multiple unit effervescent dosage form containing an acid susceptible proton pump inhibitor in the form of the racemate, an alkaline salt thereof or one of its single enantiomers or an alkaline salt thereof, and

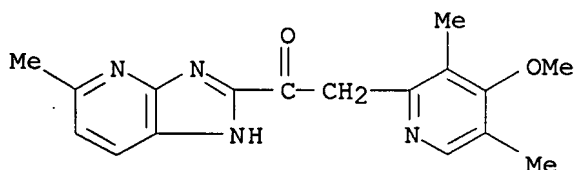
effervescent tablet constituents are claimed (Markush structure given). The proton pump inhibitor is preferably omeprazole or an alkaline salt thereof, or S-omeprazole or an alkaline salt thereof. Pellets comprising non-pareil cores 400, lansoprazole 400, hydroxypropyl Me cellulose 80, sodium lauryl sulfate 3, and water 1360 g were prepared. The above pellets (100 g) were coated with a solution comprising hydroxypropyl Me cellulose 9, polyethylene glycol-6000 1, talc 18, 95% ethanol 250, and water 250 g. The above sub-coated pellets were enteric coated with a solution comprising hydroxypropyl Me cellulose phthalate 40, acetyltributyl citrate 8, cetanol 2, 95% ethanol 162, and acetone 378 g. The enteric-coated pellets were mixed with effervescent granules (preparation given) and compressed into tablets.

IT 193335-90-9

RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(multiple unit effervescent dosage forms comprising proton pump inhibitor)

RN 193335-90-9 CAPLUS

CN Ethanone, 2-(4-methoxy-3,5-dimethyl-2-pyridinyl)-1-(5-methyl-1H-imidazo[4,5-b]pyridin-2-yl)- (9CI) (CA INDEX NAME)



=> d his

(FILE 'HOME' ENTERED AT 09:59:36 ON 30 APR 2007)

FILE 'REGISTRY' ENTERED AT 09:59:47 ON 30 APR 2007

L1 STRUCTURE UPLOADED

L2 5 S L1

L3 161 S L1 FULL

FILE 'CAPLUS' ENTERED AT 10:00:23 ON 30 APR 2007

L4 9 S L3 FULL

L5 1 S L4 AND PY<2003

FILE 'STNGUIDE' ENTERED AT 10:01:10 ON 30 APR 2007

FILE 'REGISTRY' ENTERED AT 10:02:32 ON 30 APR 2007

L6 STRUCTURE UPLOADED

L7 163 S L6 FULL

FILE 'CAPLUS' ENTERED AT 10:03:26 ON 30 APR 2007

L8 10 S L7 FULL

L9 2 S L8 AND PY<2003

=> log y

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

13.02

365.75

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE

TOTAL

ENTRY

SESSION

CA SUBSCRIBER PRICE

-1.56

-2.34

STN INTERNATIONAL LOGOFF AT 10:04:07 ON 30 APR 2007